24194 p. 10

VOLUME 6, NUMBER 8

June 1, 1970

INFORMATION INTERSCIENCE INCORPORATED

Current Awareness Bulletin

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2015043859

The following new accession from Volume 6(8) of the CURRENT AWARENESS BULLETIN may be of special interest to you.

24194 (p. 10)
CORONARY HEART DISEASE IN SEVEN COUNTRIES: SUMMARY
Keys, A.

This paper presents the summary of the findings of an international co-operative study on the epidemiology of cononary heart disease (CAD), in which international teams examined 12,700 men (11 cohorts) aged 40 through 59 years in Finland, Greece, Italy, Japan, the Netherlands, the United States, and Yugoslavia.

Data show that cigarette smoking cannot be involved as an explanation; cigarette smoking habits do not differ much between the various cohorts or cohorts combined by country.

Simon O'Shea
Project Officer
Information Storage and Retrieval System

June 1, 1970

Volume 6, Number 8 June 1, 1970

CURRENT AWARENESS BULLETIN

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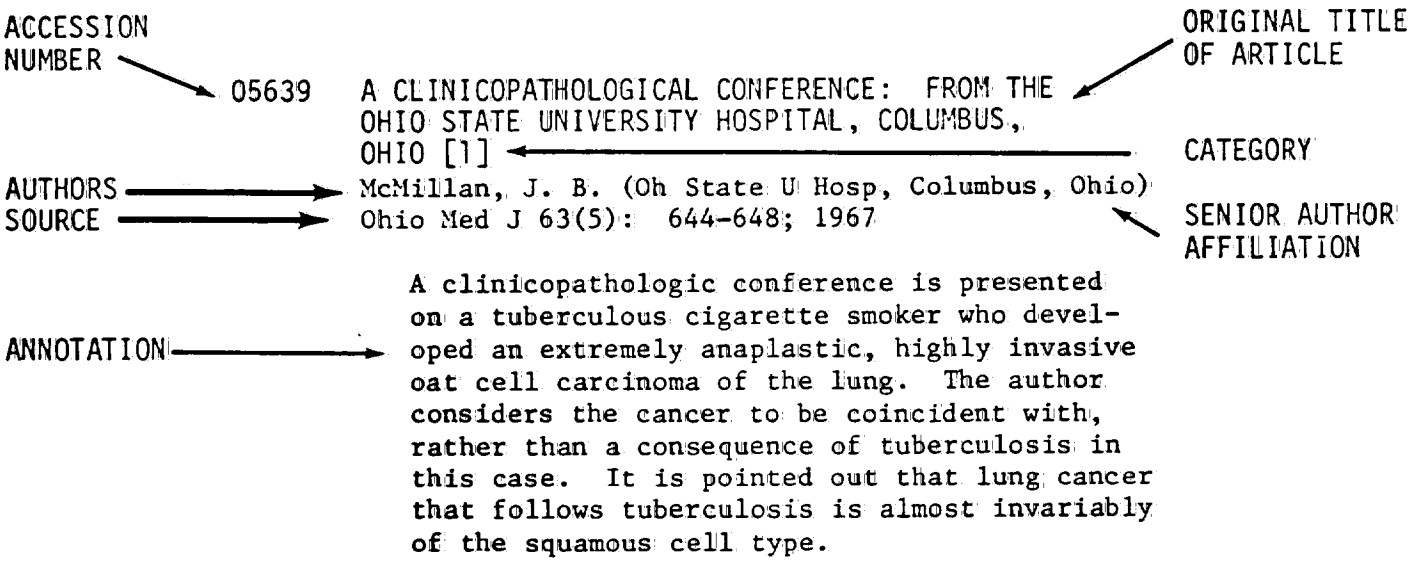

SECTION 1-CANCER

23904 MEDICINE, BIOLOGY, AND SURGERY AT THE CARLO ERBA FOUNDATION: HOW TO SMOKE WITHOUT DAMAGE: CORRELATION BETWEEN CIGARETTE SMOKING AND LUNG CANCER: INTRODUCTORY NOTES: A NEW CAUSAL ELEMENT IN THE GENESIS OF LUNG CANCER [1,4,9]

Sirtori, C. (Nat Canc I, Milan, Italy)

Med Biol Surg Carlo Erba Found: [3], 993-995; 1969

A hypothesis of lung carcinogenesis is developed, based on the production and action of tensioactive substance (surfactant).

Tensioactive substance is produced by the lamellar inclusions of the alveolar cells. These inclusions make their appearance in the fifth month of fetal life in humans. The effect on these lamellar inclusions by influenza virus, which is involved in the pathogenesis of lung cancer, was studied in Swiss mice. The first effects of exposing mice to influenza virus B Lee is a thickening of the plasma membranes of pulmonary cells, which becomes apparent at 48 hours. At 72 hours, the virus particles appear, but only in cells that have lamellar inclusions. Sometimes they are located specifically in these inclusions. Thus may be formulated the hypothesis that lesions caused by influenza virus in the lamellar inclusions reduce the availability of tensioactive substance in the lungs, thus reducing pulmonary and respiratory elasticity. The reduced elasticity would prolong the contact of lung alveoli with carcinogenic agents, such as air pollutants and cigarette smoke. "It is known that benzopyrene alone or air pollutants alone, cannot produce lung cancer: but when these substances are associated with the influenza virus, they do produce cancer of the lungs. (Presented at a Symposium on How to Smoke without Damage: Correlation between Cigarette Smoking and Lung Cancer by the Carlo Erba Foundation, held in Bologna, Italy, May 2, 1964.)

[Part of a Series: Document Nos. 23904-23905]

24130 INCIDENCE OF MULTIPLE PRIMARY CANCERS: III. CANCERS OF THE RESPIRATORY AND UPPER DIGESTIVE SYSTEM AS MULTIPLE PRIMARY CANCERS [1]

Berg, J. W./Schottenfeld, D., Ritter, F. (Nat Canc I, Bethesda, Md)

J Nat Cancer Inst 44(2): 263-274; 1970

A follow-up of 9415 patients with squamous cell carcinomas of the respiratory system, oral region, or esophagus or other cell types of lung cancer revealed that 518 later developed cancers of other sites. Only patients with cancers of the nasal cavity, paranasal sinuses, and nasopharynx did not develop excess cancers. Apparently these differences in the incidence of multiple primary cancers are due to differences in tissue susceptibility.

An excess risk of lung cancer was found among patients with primary cancers of the oral region (tongue, palate, tonsils, floor of the mouth, and pharynx) and the extrinsic and intrinsic larynx. Patients with primary oral cancer developed all histological types of lung cancer, and the percentage of squamous cell and oat cell carcinomas was similar to the relative frequency of these carcinomas in the general hospital population. Only 2 of the 36 lung cancers, however, were adenocarcinomas (instead of the six expected) while the anaplastic and other unclassified types were overrepresented.

Patients with esophageal cancer had a tenfold relative risk for new regional primary cancers. In addition to lung cancer, patients with oral cancer, particularly those with tonsillar cancer, had an excess risk of developing esophageal or laryngeal cancer. Second primary cancers occurred three times more often in the extrinsic than in the intrinsic larynx, and patients with extrinsic, but not those with intrinsic, laryngeal cancer later developed excess oral, pharyngeal, and esophageal cancer. Esophageal cancer patients had an excess risk of developing oral cancer and extrinsic larynx cancer, but not lung cancer. Lung cancer patients developed no excess esophageal cancer and only a slight excess of oral and laryngeal cancer. The three oral cancers found in lung cancer patients developed in those with squamous cell carcinomas while laryngeal cancers occurred after all histologic types except oat cell carcinoma.

The risk of a second regional cancer varied with sex and age and by site and subsite. Women were at less risk for second primary cancers than men in all but 1 of 11 subgroups. There was a trend for the risks of second cancers of the larynx and lung in men and second cancers of the esophagus and lung in women to decrease with age.

A significant excess of skin cancer was found in patients with palatine, pharyngeal, and laryngeal cancer and particularly in those with lip cancer.

A significant excess of thyroid cancer was found in patients with oral and pharyngeal cancers and a significant excess of rectal cancer in those with oral cancer, particularly in those with laryngeal cancer. A threefold excess risk of developing pancreatic cancer was seen in patients with laryngeal cancer; this excess risk is close to the 2.7-fold increase in the mortality observed in heavy smokers. Pancreatic cancer was also significantly increased after lung cancer. "Although pancreatic, kidney, and bladder cancers occur with increased frequency in heavy smokers ... pancreatic and bladder cancers were seen less often than expected in the patients of this study, whereas kidney cancer was not appreciably increased."

The excess of multiple cancers in this group of patients is believed to result from the interaction of field carcinogenesis and exposure to tobacco smoke. Medical records confirm long smoking histories among almost all patients. Alcoholism may also be a factor; particularly among women. A comparison of the data obtained in this study with that in the literature suggests, "... that most excess lung cancer was due to smoking, possibly potentiated by the additional factor of tissue susceptibility or perhaps by the additional exogenous factors ... ." Data on patients with esophageal cancer suggest that some factor of tissue susceptibility interacts with the tumorigenic activity of tobacco. Cessation of smoking is recommended as a measure which might lessen the risk of these patients' developing lung or esophageal cancer.

24241 REASON FOR OPTIMISM [1,5]
Blokhin, N. (Mosc I Exp Clin Onc, Moscow, USSR)
World Health (Feb-March): 10-13; 1970

In a question and answer session describing cancer control and therapy programs in the U.S.S.R., Dr. Blokhin states that "Tobacco is not a direct cause of lung cancer but the habit of smoking contributes to the development of precancerous conditions and subsequently of malignant tumours. Cigarettes are also an aggravating factor in cardiovascular illnesses ... ." There is a vast campaign in the U.S.S.R. to eliminate cigarette smoking and other environmental carcinogens.

Cancer of the lung is more prevalent in the U.S.A. and Western Europe than in the U.S.S.R., there is more cancer in the Southern U.S.S.R. than in the Northern U.S.S.R. and esophagus cancer is more prevalent in the Central Asian republics than elsewhere in the country.

Routine examination of the over-30 population reveals about 10/1000 with precancer, and 1/1000 with cancer.

24256 LUNG CANCER AMONG THE JEWS AND NON-JEWS OF PITTSBURG, PENNSYLVANIA, 1953-1967: MORTALITY RATES AND CIGARETTE SMOKING BEHAVIOR [1]

Herman, B./Enterline, P. E. (U Tex Med Branch, Galveston, Tex)

Amer J Epidem 91(4): 355-367; 1970

A study of lung cancer mortality (in a population aged 45 and over) by religion, in Pittsburgh, Pa., from 1953 to 1967 revealed that the lung cancer mortality rate (per 100,000 inhabitants) of the Jewish male was lower (92.5) than his non-Jewish equal (148.6), that Jewish females had a lung cancer death rate (41.8) approximately twice that for the non-Jewish females (21.7), that the low rate of lung cancer in Jewish males was due to their low cigarette consumption as compared to non-Jews, and that the high rate of lung cancer in Jewish females is not due to their cigarette smoking. As a result, the lung cancer sex ratio of Jews (2.2) was notably low when compared with the ratio of non-Jews.

The proportion of epidermoid and anaplastic carcinomas was found to be slightly lower among Jewish males relative to non-Jewish males. The category made up 82.4% of the Jewish males' lung pathology as opposed to 88.7% of those among non-Jewish males. Consequently, glandular tumors comprised a greater proportion of Jewish male histological types than was the case for non-Jewish males. The Jewish female also had a lower proportion of epidermoid and anaplastic neoplasms (63.3% versus 73.1%) and thus a higher percentage of glandular tumors contributing to her total lung pathologies compared with her non-Jewish equal. Although females tended to differ by religion in their percentage of known histopathology, it is highly unlikely that underreporting or overreporting of pathology in one religious group would lead to a bias referable to a specific type of pathology.

According to religion, the proportion of cigarette smokers was smallest among Jewish males (75.3% versus 91.8%). They also tended more to pipe and/or cigar only smoking than did the non-Jewish male (8.2% versus 3.4%). The findings for Jewish females relative to non-Jewish females also showed a greater proportion of nonsmokers of cigarettes (56.8%) than did the non-Jewish females (34.2%).

Of those male cases who smoked cigarettes, the Jews had both higher and lower proportions of light (5.5% versus 3.4%) and heavy (54.5% versus 59.0%) smoking categories, respectively. Although among female cigarette smokers, the percentage of heavy smokers by religion was basically similar (about 50%), the Jews, nevertheless, smoked less heavily, having a higher proportion of the one-to-ten-cigarettes per day category (25.0% versus 10.4%) comprising their total cigarette smoking.

Tabulated data indicate the low percentages of known information regarding duration of cigarette smoking among all groups. Nevertheless, data available were relatively consistent with those presented previously regarding type and amount of smoking. The Jewish male tended to have a slightly shorter duration of cigarette smoking than was the case for his non-Jewish peer. The Jewish female, however, appeared to have smoked for a far shorter time than did the non-Jewish female; i.e., 62.5% of Jewish females smoked less than 25 years while only 20% of the non-Jewish females did likewise. Because of the rather large discrepancy in the percentage of unknown information among the females by religion and its possible effect on the accuracy of the above findings, it was decided to estimate what the extreme effect of adding 29.2% more non-Jewish female cases to the one 25 years category would be. Although it is highly unlikely that all 11 cases would be in the same category, it was still interesting to find that, even with all of these cases placed in the shorter duration category, the new non-Jewish female value of 61.9% remained below that for the Jewish female. This figure suggests that the initial findings represented true differences between the groups.

"The present investigation demonstrated that, during the 15-year period from 1953-1967, the Jewish male had a low proportion of epidermoid and anaplastic carcinomas comprising his lung histologies relative to his non-Jewish equal. This finding suggests that the low lung cancer death rate among Jewish males might be due to their low rate of cigarette smoking. In support of such an assumption are the results regarding type, amount, and duration of smoking among the Jewish and non-Jewish male lung cancer deceased. The proportion of cigarette smokers was low for the Jews relative to the non-Jews. Furthermore, those Jewish males who did smoke, tended to smoke less heavily and for a shorter duration of time than other males. The Jews also had a higher proportion of pipe and/or cigar only smokers than did the non-Jews."

Among females who died of lung cancer during the aforementioned 15-year period, the proportion of epidermoid and anaplastic carcinomas was found lower for the Jews than for the non-Jews. This finding implies that cigarette smoking may not be the reason for the high death rate of the Jewish females relative to other females. This suggestion was supported by data on type, amount, and duration of smoking. Jewish females had a proportion of nonsmokers in excess of that found for non-Jewish females. In addition, if the Jewish female smoked, she tended to do so less heavily and for a shorter period of time than did the non-Jewish female.

Religious differences in diagnostic handling, death certificate reporting, and biological processes (heredity, constitution, and others) should be considered as possible factors influencing the lung cancer mortality patterns observed. There is also the possibility that a religious differential in frequency of metastatic (secondary) adenocarcinomas of the lung can explain the lung cancer differential between the Jewish and non-Jewish female.

"The above errors are obviously still more difficult to detect among the Jews because of their greater reluctance to undergo autopsy examination than is the case for the non-Jews. In support of such unwillingness on the part of the Jew are the findings of the present study. The Jews, both males and females, had lower proportions of cases undergoing autopsy. Among females, in the present study, the Jew had the higher proportion of ISC 163, i.e., malignant neoplasms of lung unspecified as to whether primary or secondary."

SECTION II-RESPIRATORY SYSTEM (NO CANCER)

24278 ENHANCEMENT OF RECOVERY FROM CILIASTASIS AFTER CIGARETTE SMOKE EXPOSURE BY ALUPENT^{R} (METAPROTERENOL) AEROSOL TO RABBIT TRACHEA IN-VITRO [3]

Bleiberg, M. J. (Wood Res Corp, Herndon, Virginia)
Fed Proc 29(2): [1], 242-244, 550 Abs; 1970

Metaproterenol (a bronchodilator) aerosol antagonized the ciliastatic activity of cigarette smoke on the rabbit trachea. The phenomenon took place where the bronchodilator was applied before as well as after the administration of cigarette smoke.

The present study was prompted by previously published experiments which have shown that Metaproterenol produces a sustained increased in the ciliary activity of rabbit trachea. (Presented at the 54th Annual Meeting of the Federation of American Societies for Experimental Biology, held in Atlantic City, New Jersey, April 12-17, 1970.)
[Part of a Series: Document Nos. 24275-24282]

See also: 23904, p. 1
24339, p. 8

SECTION III-CARDIOVASCULAR SYSTEM

23381 CHROMIUM, SUCROSE, AND ATHEROSCLEROSIS [6]
Altschule, M. D.
Med Count 1(9): 52; 1969

A short review discloses that feeding chromium to animals on chromium deficient diets prevented the rise in serum cholesterol associated with aging and reduced the extent of arteriosclerosis, in comparison with animals whose chromium intake was subnormal. Other studies have shown that animals fed a high sugar-low chromium diet present progressive increase in blood cholesterol levels while animals fed a high chromium-high sugar diet did not. The high cholesterol levels found in sugar-fed animals were reversed by feeding them chromium.

It is pointed out that in some highly developed countries as much as 20% of the caloric intake may be in the form of refined sugar. The reported high incidence of arteriosclerosis in populations that eat this kind of diet has stimulated interest in the low chromium content of refined sugar. A relationship between arteriosclerosis and chromium deficiency has been suggested by some. (See also Document No. 23118.)

24339 SOME ACUTE EFFECTS OF SMOKING IN SHEEP AND THEIR FETUSES [3,5]
Kirschbaum, T. H./Dilts, P. V., Brinkman, C. R. (U Cal Sch Med, Los Angeles, Calif)
Obstet Gynec 35(4): 527-536; 1970

Intravenous injection of nicotine and simulated human smoking accomplished by means of tracheal intubation failed to induce significant acute changes of the cardiovascular functions in the fetuses of pregnant ewes.

"If there are adverse effects from maternal smoking, our investigation suggests that they stem either from the chronic effects of smoking, or through the effect of variables not measured in this study. Under the conditions of this acute study, fetal protection from the effects of absorption of nicotine, smoke, and carbon monoxide appears relatively efficient."

Nicotine injection into gravid ewes resulted in a characteristic acute decrease in blood pressure followed by a more sustained prolonged blood pressure increase. Changes in uterine blood flow rates paralleled the maternal blood pressure changes accurately as described previously. In no cases were there changes in fetal heart rate, blood pressure, or umbilical vein blood flow during this time. Nicotine injection into the fetus produced a similar response in blood pressure and heart rate with parallel changes in umbilical vein blood flow rates. In general, the fetal response was abbreviated and less striking than that seen in the ewe. Further, the dose of nicotine necessary to induce changes in fetal animals was 2-3 times that necessary in maternal animals, per unit body weight.

Cigarette smoking resulted in no obvious irritation of the sheep's respiratory tract. No measurements of ventilatory characteristics in pregnant ewes were made, but there appeared a tendency for rapid shallow respiratory efforts to appear during smoking. Consumption of 1 cigarette in 1 min or less was generally followed by emesis, hypopnea, and hypotension. Generally, this response was observed through early exploratory efforts in simulating smoking. Where the ewe's entire normal tidal volume was directed through a single cigarette, the cigarette was fully smoked within 3-4 inspiratory efforts. Under these circumstances, cardiovascular deterioration resulted rapidly, and was remedied by interdiction of smoking.

The summarized results of the effects of simulated human smoking on pregnant ewes' blood gas values, blood pressure, and heart rate reflect well the changes in individual experiments and demonstrate no statistically significant changes in blood pH, blood pressure, or heart rate through the sequence of control observations, smoking, and recovery observations.

There was a tendency for reduction in umbilical vein and artery oxygen tension to parallel the maternal decrease during smoking, but these changes were not significant. Smoking was associated with no significant changes in umbilical vein blood flow rate, though there was a tendency toward an increase in some animals. The umbilical arteriovenous oxygen content difference did not change significantly nor did the rate of umbilical oxygen transfer. The ratio of uterine to fetal oxygen consumption appeared to remain roughly constant through control and smoking intervals in a single experiment.

Blood carbonmonoxyhemoglobin concentration appeared larger in fetal than in maternal blood. During smoking, there was a

significant increase in the carbonmonoxyhemoglobin concentration in the blood of the gravid ewe and a tendency to return to normal values with cessation of smoking. Simultaneously, no significant changes in fetal carbonmonoxyhemoglobin concentrations were seen.

"The validity with which the gravid sheep may be employed as a model of human smoking during pregnancy is certainly subject to question ... The experimental preparation clearly does not stimulate chronic human smoking. The effects of smoking on the observed maternal and fetal variables was generally small."

24194 CORONARY HEART DISEASE IN SEVEN COUNTRIES: SUMMARY [5,8]
Keys, A. (U Minn Sch Publ Health, Minneapolis, Minn)
Circulation 41(4, Suppl. 1): [2], I-186-I-211; 1970

This paper presents the summary of the findings of an international cooperative study on the epidemiology of coronary heart disease (CAD), in which international teams examined 12,700 men (11 cohorts) aged 40 through 59 years in Finland, Greece, Italy, Japan, the Netherlands, the United States, and Yugoslavia.

Great differences between cohorts in age-standardized prevalence rate of CAD were observed at entry, electrocardiographic (ECG) evidence of old myocardial infarction being many times higher in the U.S. and in Finland than in Yugoslavia, Greece, Italy, and Japan. Angina pectoris and CAD diagnosed on "softer" clinical and ECG criteria tended to show similar population differences. The prevalence rates of CAD of the cohorts tended to be directly related to characteristics of the cohorts in regard to blood pressure and serum cholesterol but not in regard to relative body weight or body fatness or smoking habits of the cohorts.

During five years there were 588 deaths in the entire study population, 158 from CAD. Among the U.S. railroad men 62 of 125 deaths were due to CAD; in Finland 38 of 111 deaths were due to CAD; in the Netherlands 16 out of 50 deaths were thus accounted for. For all other cohorts combined, only one out of eight deaths was due to CAD. That low proportion did not reflect a high death rate ascribed to other, non-CAD causes.

In most of the cohorts, low CAD death rates were associated with low all-causes deaths, the standard basis of comparison for CAD and all-causes deaths being the deaths expected to match the five-year experience of equal numbers of white men in the United States with the same age distribution.

Among U.S. railroad men age-standardized death rates from all causes were higher among switchmen (physically active)

than men in sedentary occupations while the reverse was true of CAD deaths, but in neither case was the difference statistically significant.

Among 12,529 men judged to be free from CAD at the entry examination, the five-year CAD experience, in a hierarchy of mutually exclusive diagnostic categories, was as follows: 116 deaths from CAD, 113 nonfatal definite myocardial infarctions, 219 cases of classic angina pectoris, and 113 men given the diagnosis of CAD on the basis of less rigid and specific clinical and ECG criteria. The crude average annual rate for all CAD incidence in these men initially CAD-free was 102.3 per 10,000. For CAD incidence meeting the hardest criteria--deaths and definite infarcts--the corresponding rate was 36.5 per 10,000 men.

The age-standardized CAD incidence rate for men CAD-free at entry differed greatly in the several cohorts, the extremes being in Finland and the U.S. on the high side with the cohorts in Japan, Greece, and Yugoslavia on the low side. Within countries there were no significant differences between cohorts in CAD rates except for rural Finland where the total CAD incidence rate in the east was definitely higher than in the west.

In general, the several categories of CAD diagnosis tended to show much the same picture for differences in rates between cohorts, but the rates for angina pectoris were not in close conformity with the rates for the objective and standardized diagnoses of CAD death and nonfatal infarction. In the Netherlands angina pectoris apparently contributed a lower proportion of total CAD incidence than in the other areas; there is no way of deciding whether this apparent peculiarity reflects a difference in the manifestation of the disease or diagnostic conservatism.

Examination of the representation in the several cohorts of the so-called risk factors shows that most of those factors, whatever may be their influence within cohorts, cannot explain the observed differences in the incidence of CAD. Data show that cigarette smoking cannot be involved as an explanation; cigarette smoking habits do not differ much between the various cohorts or cohorts combined by country.

Differences between the cohorts in the proportion of the men who are sedentary or physically inactive do not explain the differences between the cohorts in the incidence of CAD.

There were large differences between cohorts in body fatness and relative body weight, but consideration of neither obesity nor relative weight helps to explain the population differences in CAD incidence. There was some tendency for the incidence of CAD to be related to the prevalence of hypertension in the cohorts; at least hypertension was less common in the cohorts with the lowest incidence of CAD. "It is not possible, of

course, to insist that the blood pressures recorded for the several cohorts are strictly comparable, especially for diastolic pressure. Common instructions were issued about recording blood pressure, but uncertainties remain in the absence of identity of the environment and of a measuring device without human intervention. The role of blood pressure in CAD incidence is more reliably examined within cohorts."

The CAD incidence rates of the cohorts were just as closely related to the dietary saturated fat as to the serum cholesterol level. Serum cholesterol averages and CAD incidence rates were not found to be related to the percentage of calories provided by protein or polyunsaturated fatty acids in the diet and were only slightly related to total fat calories.

The analysis of the relationships within cohorts consistently indicated the importance of blood pressure and serum cholesterol. When "hard" criteria of CAD death and infarction were used for diagnosis, CAD incidence was not significantly related to either relative body weight or to body fatness. When all CAD diagnoses were used in computing incidence rate, there was a weak tendency for the rate to be related to relative body weight as well as to body fatness. That tendency was not statistically significant when the confounding influence of blood pressure was removed.

CAD incidence was significantly related to smoking habits in the U.S. railroad men but not in the European cohorts. All-causes death rate was also related to smoking habits in the U.S. railroad men but not in the other cohorts.

No significant relationship between habitual physical activity and any measure of CAD incidence rate were found. If there were a true excess of 15 to 20% CAD among sedentary men, the present material would be too small to prove it.

A significant relationship was found between the presence of certain ECG abnormalities at entry and the future occurrence of CAD.

The coefficients for the multiple logistic equation for CAD risk, obtained by others from Framingham data, were tested with the present material. Comparison of absolute numbers of CAD cases with those "predicted" was considered to be improper because of lack of identity in the diagnostic methods and the question of comparing five-year observed rates with predictions based on 12 years of follow-up. Analysis in terms of ratios of rates would seem to be reasonable to consider, however. When both observed and predicted CAD incidence rates for the various cohorts were expressed as ratios of the observed and predicted rates of the U.S. railroad men, the correspondence between observed and predicted ratios was unexpectedly good.

There was no indication that the incidence of CAD was inversely related to the incidence of any other disease or that, in effect, rarity of CAD in a cohort was compensated by an excess

of other affliction. In general, the total all-causes death rate reflected the death rate from CAD with the result that the all-causes death rate was remarkably low in several of the cohorts with the lowest incidence of CAD. In Slavonia, an apparent exception, unusual causes of death were involved.

In spite of the bad experience in Slavonia, for all five Yugoslav cohorts combined, all-causes death totaled 123 compared with 175.5 expected for age-matched white men in the United States, O/E (observed/expected)=0.70. The all-causes mortality of the three Italian cohorts was also favorable with O/E=0.80 (114 deaths observed, 142.3 expected to match U.S. white men), as was that of the Japanese men with O/E=0.78 (47 deaths, 60.3 expected). The Greeks had by far the best experience with only 21 deaths instead of 70.5 expected, O/E=0.30. CAD was relatively much less common in all of these cohorts than in the Finns, the men of Zutphen, and the U.S. railroad men.
[Part of a Series: Document Nos. 24179-24194]

See also: 23904, p. 1
24241, p. 3

SECTION IV-MISCELLANEOUS

24253 INVASION OF PRIVACY: NEW ANGLE ON SMOKING [7,9]
Kennedy, R. B. (Miss St Med Ass, Jackson, Miss)
J Mississippi Med Ass 11(3): 117-118; 1970

An editorial reviews "... signs of the times catching up to the long suffering of nonsmokers caught in a tobacco smog.": nonsmoking sections of Pan Am's 747 jumbo jet; smoking restriction at Presidential press conferences; Ralph Nader's efforts to prohibit smoking in airplanes, buses, hospitals, and public accommodations; the order by the U.S. Air Force Surgeon General prohibiting smoking by hospital patients and forbidding hospital cigarette sales; the Mississippi State Medical Association's prohibition of cigarette sales at their headquarters; efforts for equal time and space for antismoking propaganda; the formation of Citizens to Restrict Air Line Smoking Hazards (CRASH); the letter from Chief Justice Warren Burger to protest smoking on airplanes; and the possibility that new electronic controls on airplanes are adversely affected by tobacco tar.

Italian investigators claim that sidestream cigarette smoke may constitute a health hazard to nearby nonsmokers. The mean burning time of a cigarette is 12 minutes; it is inhaled for only 24 seconds, leaving more than 11 minutes of sidestream smoke for the distress of nonsmokers.

The new militant antismoking movement has legal and medical overtones. Some individuals are allergic to tobacco smoke and nearly all nonsmokers find it offensive. It is contended in some legal circles that uncontrolled smoking constitutes an invasion of privacy. "The smoker has no particular claim on anybody else's airspace, and the fact of the matter is that man's natural state is not to smoke. ...

We wish smokers no ill, for they have enough woe already. We simply wish nonsmokers well with as much fresh air as in currently available. In fact, common courtesy should make laws, regulations, and lawsuits quite unnecessary as America slowly comes to its senses about tobacco and health."

24244 THE HABIT OF SMOKING MAY BE PHYSIOLOGICALLY REINFORCED [7,9]
Anonymous
Chem Eng News 48(18): 65; 1970

ac-cine 11/27/72 Bhagat

A news report states that, "The habit of smoking may be physiologically reinforced and may get an assist from drinking ...."

Experiments on rats show that nicotine injections greatly increase production of norepinephrine, which stimulates the emotions. A smoker with high norepinephrine levels induced by nicotine would need habitual doses of nicotine to avoid depression which accompanies reduction of norepinephrine.

Since alcohol reduces norepinephrine levels, it has been suggested that, "... an individual who imbibes large quantities of alcohol would have to smoke more to compensate for the effect of the alcohol."

24287 CIGARETTE SMOKING AND BODY FORM IN PEPTIC ULCER [7]
Monson, R. R. (Harv U Sch Publ Health, Boston, Mass)
Gastroenterology 58(3): 337-344; 1970

A questionnaire study of 645 physicians of both sexes with peptic ulcer and 621 controls revealed that physicians with gastric or duodenal ulcers have smoked more than comparable control subjects. Those with duodenal ulcer started smoking at an earlier age than did control subjects. Among those physicians who smoked cigarettes, however, patients and control subjects did not differ in percentage who had stopped, years smoked, or number of packs smoked per day. It is suggested that cigarette smoking and peptic ulcers result from a common cause; the role of smoking in the etiology of ulcer must be questioned. "That there is an association between cigarette smoking and peptic ulcer cannot be doubted. In all studies, smokers have more ulcers than nonsmokers. Further, among ulcer patients, those who smoke have a higher rate of morbidity and mortality than those who do not. But the question still remains as to whether smoking is a cause of ulcer.

"It might be supposed that, if smoking were a cause, heavier smokers would be at greater risk. The study of Doll et al. and the present study have not found an excess of heavy smokers in those with ulcer ....

"The finding that physicians with duodenal ulcer started smoking at an earlier age than control subjects, but did not smoke longer or stop less often, adds credence to the theory that cigarette smoking is not a direct cause of duodenal ulcer. It seems more likely that whatever causes a person to smoke also is important in the future development of an ulcer.

"Since there were so few physicians with gastric ulcer, it is difficult to differentiate between their smoking habits and those of physicians with duodenal ulcers."

Body form as measured by height, weight, and ponderal index was compared between smokers and nonsmokers as well as between patients and control subjects. These comparisons were made for age 21 and for the time of the study in 1968. Physicians with duodenal ulcer were more slender than controls in 1968, but did not differ at age 21. The reported linearity of persons with duodenal ulcer therefore may be secondary to the ulcer rather than associated directly with a cause of ulcer. Physicians with gastric ulcer did not differ from controls. No association between body form and cigarette smoking could be demonstrated among duodenal ulcer patients; control smokers as well as all smokers were significantly more slender than nonsmokers in 1968 but not at age 21.

See also: 24194, p. 10

SECTION V-MEDICAL OPINION

See 23904, p. 1
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